Recovery from ECS-produced amnesia*

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Sixty-four naive male albino rats were trained to leverpress for sucrose using a discrete trial procedure. Half of the Ss were trained on a continuous reinforcement schedule (CRF) and the other half were trained on a fixed ratio schedule (FR). Following acquisition, for half of the Ss in each group a leverpress produced footshock followed by ECS, and for the remaining Ss a leverpress produced footshock only. Half of the Ss in each group were tested 24 h after treatment; for the remaining Ss testing was delayed for 10 days. Results showed that suppression of the leverpress response due to footshock was reduced by the ECS, regardless of acquisition reinforcement schedule, for Ss tested 24 h after treatment. For Ss tested 10 days after treatment, there was apparent spontaneous recovery of the conditioned emotional response (CER), since there were no differences between the shock-only and shock-plus-ECS groups. Results further showed no recovery of the partial reinforcement effect (PRE).

The administration of electroconvulsive shock (ECS) following a one-trial learning experience appears to produce retrograde amnesia (RA) for that learning experience (Lewis, 1969; Spevack & Suboski (1969). This phenomenon has been explained by a memory consolidation hypothesis which suggests that following sensory input, a physiological consolidation process, operating over time, permanently fixes the memory trace. It is assumed that during the consolidation process the memory trace is relatively unstable and susceptible to disruption (McGaugh, 1966). Presumably, then, the massive electrical interference produced by ECS disrupts the ongoing consolidation process, resulting in the destruction of traces not yet fixed, but has no effect on traces already permanently fixed.

A typical procedure is to administer aversive stimulation, such as footshock, upon the emission of a previously learned response. The aversive stimulation results in a suppressed rate of responding; however, if ECS is administered shortly after the aversive stimulation, response suppression does not occur, indicating RA for this one-trial learning.

A number of recent studies (Cooper & Koppenaal, 1964; Kohlenberg & Trabasso, 1968) have shown spontaneous recovery over time from ECS-produced amnesia, the results of which tend to weaken a memory consolidation hypothesis. If ECS destroys the memory trace which is in the process of becoming permanently fixed, that trace should be permanently lost. Although negative results have been reported (Chevalier, 1965; Greenough, Schwitzgebel, &

Fulcher, 1968), spontaneous recovery is reported sufficiently often to presumably worry a memory consolidation theorist.

More recently, Young & Galluscio (1970a) reported findings that would seem to further weaken a consolidation of memory hypothesis. In that study two groups of rats were trained to leverpress, one group on a continuous reinforcement schedule (CRF) and the other on a fixed ratio schedule (FR 5). Upon completion of acquisition, on pretest day, a leverpress was followed by footshock. For half the Ss in each group, footshock was followed by ECS; the other half received no ECS. All Ss were then tested during an extinction session. Results showed that during extinction the CRF group which received ECS following footshock made significantly more responses than did the CRF group which received footshock only. However, both FR 5 groups showed significantly suppressed response rates, not significantly different from each other, although one group had received footshock plus ECS and the other group had received footshock only. Thus ECS did not eliminate suppression of response rate following training on a FR schedule.

To determine if acquisition rate is a critical variable, Young & Galluscio (1970b) replicated their first experiment; however, in the second experiment a discrete trial procedure was used to control response rates. With this technique the acquisition response rate for FR-trained Ss was significantly lower than that for CRF-trained Ss, and results showed that ECS apparently produced RA in both groups. This suggests that the effect of ECS may be a function of acquisition response rates. A second finding of the Young and Galluscio studies was the failure of FR-trained

Ss to show the partial reinforcement effect (PRE)—the increased resistance to extinction shown by Ss trained on a partial reinforcement schedule.

In a similar study, Young & Day (1970) reported the failure of Ss trained on a variable ratio (VR) schedule to show the PRE following the administration of ECS.

The present study was designed to replicate the Young & Galluscio (1970b) experiment, which showed that ECS produced RA in FR-trained Ss when acquisition response rate was low, and to determine: (1) if there was spontaneous recovery from ECS-produced RA over time and, if so, (2) if there was spontaneous recovery of the PRE over time. If the conditioned emotional response, produced by aversive stimulation and for which ECS produces RA, spontaneously recovers over time (Miller, 1968), then perhaps this recovery would be accompanied by recovery of the PRE.

SUBJECTS

The Ss were 64 naive male albino rats, 175-200 g in weight at the start of the experiment.

APPARATUS

The apparatus consisted of two identical Scientific Prototype operant chambers, each enclosed in a sound-insulated, ventilated cubicle. Each operant chamber had a grid floor, and fitted on the end wall was a retractable lever and a liquid dipper that dispensed .01 ml of a 40% sucrose solution, used as reinforcement. All E-controlled events were operated by an electronic programming device.

PROCEDURE

The Ss were selected randomly from the LSU colony, placed in individual cages and kept on an ad lib food and water schedule for 4 days, after which Ss were placed on a food-deprivation schedule of 10 g of Purina Chow every 24 h. Water was available in the cages at all times, and Ss were fed approximately 10 min after each experimental session.

From the 5th to the 9th day, Ss were handled in pairs for 5 min daily, and on the 10th day, Ss began magazine training on a VI 30-sec schedule. Experimental periods consisted of 20 presentations of the dipper and were continued for 4 days. On the 14th day, all Ss were conditioned to leverpress and on the following day were allowed to make 100 reinforced responses. On the 16th day, Ss were divided randomly into two groups, FR and CR, and began acquisition training. For the FR group, acquisition was under a FR 2 reinforcement schedule for the first 5 days and under FR 3 for the last 5 days. For the CR group, acquisition was under a CRF schedule throughout.

*This research was supported in part by a grant from the University Council on Research, LSU, to the first author.

Table 1
Mean Number of Leverpresses in Extinction

Group	First Minute	Total	Group	First Minute	Total
CR (ECS)	7.75	53.50	CRD (ECS)	6.37	43.50
CR (SO)	2.87	34.50	CRD (SO)	3.62	40.87
FR (ECS)	7.25	57.00	FRD (ECS)	4.25	33.37
FR (SO)	2.62	36.87	FRD (SO)	1.50	29.00

Table 2
Mean Number of Responses Per Minute

Group	Acquisition	Testing	Group	Acquisition	Testing
CR (ECS)	34.63	5.35	CRD (ECS)	36.36	4.35
CR (SO)	36.23	3.45	CRD (SO)	37.31	4.09
FR (ECS)	15.31	5.70	FRD (ECS)	18.18	3.34
FR (SO)	16.08	3.69	FRD (SO)	14.53	2.90

All Ss were given 100 leverpresses daily for 10 days. A discrete trial procedure was used and the lever, which required 4 sec to retract and extend fully, was inoperative during retraction.

On the day following completion of acquisition, each group was subdivided randomly, resulting in the following eight groups ($N = 8$):

Groups CR(ECS) and FR(ECS)

On treatment day, Ss in these two groups were fitted with a harness. Fine wire from an ECS source entered the operant chamber from the top, was connected to the harness, and from the harness to microalligator clips which attached to S's ears. The harness arrangement permitted complete freedom of movement to all parts of the operant chamber. The first leverpress produced a 9-mA footshock of 2 sec duration, delivered through the grid floor, and the lever retracted. The offset of footshock initiated the onset of a 50-mA 500-msec ECS delivered through the ear-clip electrodes.

Groups CR(SO) and FR(SO)

For Ss in these two groups, treatment was identical to the two groups above except that footshock was not followed by ECS.

Groups CRD(ECS) and FRD(ECS)

For Ss in these two groups treatment was identical to that given to the CR(ECS) and FR(ECS) groups, but testing was delayed for 10 days following treatment.

Groups CRD(SO) and FRD(SO)

For Ss in these two groups treatment was identical to that given to the CR(SO) and FR(SO) groups, but testing was delayed for 10 days following treatment.

For all groups testing consisted of a 10-min extinction session with the no-delay groups tested 24 h after treatment and the delay groups tested 10 days after treatment. During extinction the stimulus conditions were the same as during acquisition except that the dipper was inoperative.

RESULTS

The number of responses made by each S in extinction was recorded at the end of the first minute and results showed that more responses were made by Group CR(ECS), followed in order by Groups FR(ECS), CRD(ECS), FRD(ECS), CRD(SO), CR(SO), FR(SO), and FRD(SO). These data were subjected to an analysis of variance which showed that only the treatment effect was significant ($p < .01$). Group comparisons (t tests) showed CR(ECS) vs CR(SO) and FR(ECS) vs FR(SO) significant beyond the .01 level. The comparisons of CRD(ECS) vs CRD(SO) and FRD(ECS) vs FRD(SO) were not significant at the .05 level.

The total number of responses made by each S for the 10-min period showed that more responses were made by Group FR(ECS), followed in order by Groups CR(ECS), CRD(ECS), CRD(SO), FR(SO), CR(SO), FRD(ECS), and FRD(SO). These data were subjected to an analysis of variance which showed that the main effect of delay was significant beyond the .05 level and that the main effect of treatment was significant beyond the .01 level; however, the main effect of schedule was not significant. The Delay by Treatment interaction was significant beyond the .05 level, and these data were therefore subjected to an analysis of variance for simple effects. The results showed that the effect of delay was not significant for the SO groups; however, the effect of delay was significant beyond the .01 level for the ECS groups. Results also showed that the treatment effect was significant beyond the .01 level for the no-delay Ss, but was not significant for Ss which received delayed testing. Group comparisons (t tests) showed that the comparison FR(ECS) vs FRD(ECS) was significant beyond the .01 level, but CR(ECS) vs CRD(ECS) was not significant at the .05 level. Further comparisons showed that

CR(ECS) vs CR(SO), and FR(ECS) vs FR(SO) both were significant beyond the .05 level. The mean number of responses in extinction for all groups is shown in Table 1.

Table 2 shows group mean response rates on the last 2 days of acquisition and during the test session. Acquisition response rates were significantly lower for FR-trained Ss than for CR-trained Ss ($p < .001$, Mann-Whitney U test). A comparison of response rates during testing showed Group FR(SO) to be significantly slower than Group FR(ECS), $p < .05$; Group CR(SO) to be significantly slower than Group CR(ECS), $p < .05$; and Group FRD(ECS) to be significantly slower than Group FR(ECS), $p < .05$ (Mann-Whitney U tests).

DISCUSSION

The finding that for Ss tested without delay, the ECS groups made significantly more responses than did the SO groups, regardless of reinforcement schedule, replicates the findings of Young & Galluccio (1970b).

The most critical finding of this study is that when testing was delayed for 10 days after completion of acquisition and treatment there were no differences between the ECS and SO groups. Examination of the data shows that this was due to a decline in responding for the delayed-ECS groups. In fact, the mean number of responses made by the SO and the delayed-SO groups were almost identical, whereas the delayed-ECS groups made significantly fewer responses than did the ECS groups that experienced no delay.

These data are in agreement with previous findings (Kohlenberg & Trabasso, 1968; Cooper & Koppelaar, 1964) and suggest that the conditioned emotional response (CER) due to aversive stimulation may be temporarily suppressed by the administration of ECS and spontaneously recover over time. If such recovery does occur, however, it is not accompanied by recovery of the PRE. In fact, the decline in responding for the FRD(ECS) group was *greater* than that for the CRD(ECS) group. If the PRE is caused by some type of conditioned emotional response, such as frustration (Amsel, 1958), it may be that spontaneous recovery of the CER due to aversive stimulation interferes in some way with the frustration response or its connection with the stimuli of the experimental situation, resulting in a loss of the PRE.

An alternative hypothesis would suggest that the net effect of the administration of ECS is an inflated rate of responding, perhaps due to

disinhibition; that this effect is temporary and dissipates with time; that ECS either neutralizes the emotional response which causes the PRE or interferes with the previously established connection between the emotional response and the stimuli of the experimental environment.

Regardless of interpretation, however, these findings do not support the hypothesis that memory consolidates over time and that the effect of ECS is the disruption of a memory trace that is in the process of becoming permanently fixed.

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may be a valuable new tool in the study of memory through amnesia, especially when used with those drugs known to retard the different manifestations of oxygen toxicity (Johnson & Bean, 1954; Taylor, 1956; Wood et al, 1967). The first study reported here is a replication of Bean's earlier observation, and the second tests the generality of the retention deficit.

APPARATUS

A small animal pressure chamber, approximately 23 in. long and 15 in. in diam and with a Plexiglas window for observation, was used for the oxygen exposure. The chamber contained a small cage for restraining the animal and two packages of baralyme to aid in the control of the CO₂ level in the chamber. Pressurizing the chamber with oxygen was accomplished by means of a high-pressure oxygen cylinder and regulator.

EXPERIMENT 1

In a replication of Bean's earlier work, six adult rats (Wistar) were placed on a 23-h food-deprivation schedule and maintained at 80% of their original weight. Each S was given one 75-mg Noyes food pellet in the goalbox of a Lashley III maze (1964) before regular feeding once a day for 6 days prior to training. All animals were trained to a criterion of 9 out of 10 errorless trials with retracing errors prevented by guillotine doors. Trials were presented in blocks of 15 per day, except for the first and second days of training, on which the animals received 5 and 10 trials, respectively. The order in which the animals were trained was reversed from day to day. The experimental and control groups were matched in terms of the number of errors required to learn the maze.

Twenty-four hours after the last training session, the experimental animals received OHP twice a day for 10 days. The animals were sealed one at a time into the chamber, which was then flushed for 3 min with oxygen at a flow rate of 50 to 65 liters/min. The pressure inside the chamber was raised to 60 psig over a 4-min period (1 at/min). The S remained at pressure for 15 min or until it showed signs of convulsions (twitching), whichever came first. The ventilatory flow rate of the chamber during this period was approximately 40 liters/min. Gas analysis of the chamber at the end of the 15-min period at 60 psig was 0.0% CO₂, 0.8% N₂, and 99.2% O₂ at 25°C.

Twenty-four hours after the last exposure to OHP, the Ss were retrained in the maze to the original criterion. The control animals were left in their home cages, except for daily weighing, for the 10 days prior to retesting.

The effect of hyperbaric oxygen on memory*

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The effect of oxygen at high pressure (OHP) on memory was investigated. Multiple exposures to OHP after training in a Lashley III maze produced significant retention deficits, confirming a previous study. However, exposures to OHP failed to produce retention deficits of a T-maze position habit. The results suggest that the Lashley III maze deficit was produced by alterations in activation and not by a memory deficiency.

That oxygen at high pressure (OHP) causes oxygen toxicity resulting in convulsions of the grand mal type has been known since the classical work of Bert in 1878. Subsequent research has demonstrated that cellular metabolism, especially that of the central nervous system, is markedly depressed by exposures to OHP (Dickens, 1946; Haugaard, 1968). The posttrial administration of convulsive

seizures (by electroconvulsive shock) and depression of central nervous system metabolism (by drugs) have been found to be effective in producing retrograde amnesia (Chorover & Schiller, 1965; McGaugh & Petrinovitch, 1965). Consequently, it might be expected that OHP would be an effective amnesic agent. Bean and his associates (1943, 1945) have, in fact, reported that daily exposures to OHP after training in a Lashley III maze produced a retention deficit, as measured both by errors and trials to criterion during retraining.

In view of the increasing use of OHP as a medical therapeutic, elaboration of these findings should be of considerable interest to medical personnel conducting high-pressure oxygen therapy. Furthermore, OHP

*This research was supported by Grants MH10320 and NS07261 from the United States Public Health Service. We would also like to acknowledge the invaluable assistance of David Desautels, Chief of Inhalation Therapy, Shands Teaching Hospital, University of Florida, in the design of the hyperbaric chamber used in this research.